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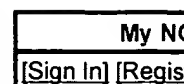
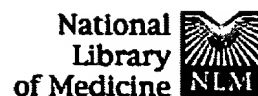
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Synuclein proteins and Alzheimer's disease.

Brookes AJ, St Clair D.

MRC Human Genetics Unit, Western General Hospital, Edinburgh, UK.

In Alzheimer's disease, synuclein/NAC (non-amyloid beta component of Alzheimer's disease amyloid) proteins are found in presynaptic cholinergic nerve terminals that degenerate early in Alzheimer's disease, and they are also found closely linked to beta-amyloid fibrils in senile plaques. Synuclein/NAC proteins provide a potential molecular link between the degeneration of cholinergic nerve terminals, and the formation of plaques, and might have a primary role in their development.

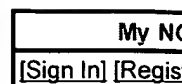
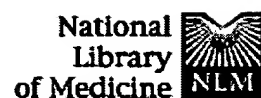
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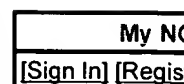
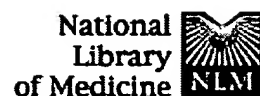
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FULL-TEXT ARTICLE**The synucleins: a family of proteins involved in synaptic function, plasticity, neurodegeneration and disease.****Clayton DF, George JM.**

Beckman Institute and Dept of Cell and Structural Biology, The University of Illinois, Urbana 61801, USA.

Synuclein proteins are produced, in vertebrates, by three genes. They share structural resemblance to apolipoproteins, but are abundant in the neuronal cytosol and present in enriched amounts at presynaptic terminals. Synucleins have been specifically implicated in three diseases: Alzheimer's (AD), Parkinson's (PD) and breast cancer. In AD, a peptide derived from alpha-synuclein forms an intrinsic component of plaque amyloid. In PD, an alpha-synuclein allele is genetically linked to several independent familial cases, and the protein appears to accumulate in Lewy bodies. In breast cancer, increased expression of gamma-synuclein correlates with disease progression. In songbirds, alpha-synuclein expression is correlated with plasticity in the developing song control system. Although the normal function of synucleins is unknown, a role in membrane plasticity seems likely.

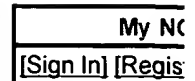
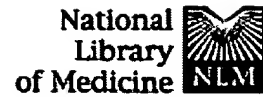
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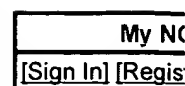
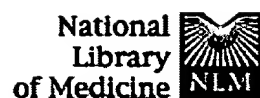


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**Immunological approaches as therapy for Alzheimer's disease.****Solomon B.**

Department of Molecular Microbiology & Biotechnology, George S. Wise
Faculty of Life Sciences, Tel-Aviv University, Ramat Aviv, Tel-Aviv
69978, Israel. beka@post.tau.ac.il

The pathology of Alzheimer's disease (AD) shows a significant correlation between beta-amyloid peptide (AbetaP) conformation and the clinical severity of dementia. For many years, efforts have been focused on the development of inhibitors of beta-amyloid (Abeta) formation and its related neurotoxic effects. The author has developed a new concept showing that site-directed antibodies may modulate formation of Abeta. The performance of anti-Abeta antibodies in transgenic mice models of AD showed that they are delivered to the central nervous system (CNS), preventing in vivo formation of Abeta. Moreover, these antibodies dissolve Abeta plaques and protect the mice from learning difficulties and age-related memory deficits. Experimental active immunisation with Abeta (1-42) in humans has been stopped in Phase II of their clinical trials. However, several new preparations, able to provide antibodies against Abeta by either active or passive routes, have been formulated and at least one of these is likely to reach clinical testing. These data support the hypothesis that AbetaP plays a central role in AD and antibodies which modulate Abeta conformation may lead to immunotherapy of the disease.

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